Comment on: Delamanid susceptibility testing of Mycobacterium tuberculosis using the resazurin microtitre assay and the BACTEC™ MGIT™ 960 system

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already built rather than starting anew. The simplicity of naming resistance gene determinants will not only help in maintaining the interest of early-career academics and clinicians in understanding antimicrobial resistance determinants, but also encourage more research into the evolution of resistance genes.

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This letter is dedicated to the hard work of Karen Bush and George Jacoby and their team, who have worked relentlessly in assigning numbers to β-lactamase variants since 1997.

Transparency declarations

None to declare.

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Comment on: Delamanid susceptibility testing of *Mycobacterium tuberculosis* using the resazurin microtitre assay and the BACTEC™ MGIT™ 960 system

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Sir,

Schena *et al*.[1] recently reported a BACTEC™ MGIT™-based protocol for delamanid drug susceptibility testing (DST). While we appreciate the authors' carefully and thoroughly executed work, we would like to take the opportunity to briefly comment.

Schena *et al*.[1] criticize a previously established protocol as follows: 'Recently, Keller *et al*.[2] proposed a DST protocol for delamanid in the MGIT, but the study had two major weaknesses: (i) the sample size of strains (25 *M. tuberculosis* isolates) was rather small; and (ii) the authors used delamanid derived from mortared pills instead of pure substance, as recommended by WHO.[3]' The recommendation Schena *et al*.[1] refer to is not an official statement by the WHO, but a publication by Diacon *et al*.[3] As pointed out by Keller *et al*.,[2] it was impossible to obtain delamanid from the producer, Otsuka, without unacceptable binding conditions. Keller *et al*.[2] readily addressed the limitations of their study: 'Further studies evaluating *in vitro* laboratory drug MICs using pure compounds and PK/PD and clinical data from a large number of drug-susceptible and drug-resistant strains are required to define clinical breakpoints.' Most notably, the epidemiological cut-off determined by Schena *et al*.[1] (0.06 mg/L) is virtually identical to the epidemiological cut-off determined by Keller *et al*.[2] (0.04 mg/L). The small difference likely reflects the different 2-fold dilution series.

Experiments conducted in our laboratory indicate that the use of pure delamanid may be problematic, as the drug appears somewhat unstable during incubation in growth medium. Thus, we are wondering whether the substance Schena *et al*.[1] received from Otsuka for their study is indeed pure delamanid, as stated in the Materials and methods section, or whether a stabilizer had been added by the producer.

We are surprised that Schena *et al*.[1] are not concerned by the drug company's policy of denying access to delamanid without extensive binding conditions for use and data publication. This is a policy not previously seen for antimicrobials entering the market, as antibiotic susceptibility testing (AST) needs to be established and verified independently by expert laboratories. Rather, Schena *et al*.[1] criticize our work, which we did to avoid the restrictions imposed by Otsuka and to develop expert AST independently of company interests.

Transparency declarations

None to declare.

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